the necrotic core. It was suggested that patients with plaques exhibiting these characteristics might benefit from selective use of distal protection devices. Neither study suggested that a relationship exists between original plaque volume and the occurrence of embolization.

In our opinion, these studies have a number of limitations. Kawaguchi et al. (1) chose ST-segment re-elevation immediately after stent implantation as a proxy for embolization. This parameter has not previously been linked to the more recognized end points of myocardial blush grade or 90-min ST-segment resolution (3). Additionally, the limited ability of the 20-MHz Eagle Eye IVUS catheter (Volcano Therapeutics, San Diego, California) to identify luminal thrombus may be particularly relevant in their patients who received only aspirin as a procedural antiplatelet therapy. Moreover, the possibility of side branch occlusion during stent deployment as a cause of myocardial necrosis does not appear to have been considered. In patients with stable angina, Kawamoto et al. (2) suggested that plaques with larger necrotic cores were more likely to exhibit high-intensity transient signals detected by a Doppler guidewire. Histopathological correlates between VH-IVUS and pathology are questionable, and neither study uses post-procedure IVUS imaging of residual plaque. Using 40-MHz IVUS imaging, a direct relationship has been demonstrated between the change in plaque volume and evidence of new myocardial necrosis (4). Thus, although it is perhaps intuitive that friable plaque elements are more likely to be liberated than are more readily compressed fibrotic components, further investigation is required.

In the accompanying editorial, Shah (5) questions whether no reflow is merely a marker of myocardial injury or whether it is the cause of further myocardial damage. There is currently no data to support the notion that, in established no reflow, administration of any pharmacologic agent has any impact on subsequent prognosis, and perfusion in areas of new myocardial necrosis remains impaired for at least 24 h despite normal perfusion in viable areas of myocardium subtended by the same vessel (6). These data suggest that no reflow is predominantly a marker of downstream myocardial necrosis. As more diffuse and challenging diseases are being tackled by percutaneous coronary intervention (PCI), further research into the prevention of plaque embolization is essential to allow further improvement in PCI outcomes.

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Reply

We appreciate the interest in our recent study (1) by Dr. Porto and colleagues. Myocardial blush grade (MBG) and early ST-segment resolution are well-known parameters used for describing the effectiveness of myocardial reperfusion, which is an independent predictor of long-term mortality (2). However, the aim of our study was to investigate distal embolization that occurs immediately after stent implantation: in other words, distal embolization induced by stent implantation. Although stent-induced distal embolization is one of the causes of low blush grade or early ST-segment resolution, it is not the only cause—several factors such as vasoconstriction, additional pharmacologic therapy, and time interval after stent implantation may also affect the outcome of these parameters. Because we were not investigating final coronary flow and prognosis, we believed that we did not need to consider the relationship between ST-segment re-elevation (STR) and MBG or early ST-segment resolution. Thus far, we have not come across parameters specific to estimating the extent of stent-induced distal embolization, and certainly, as we mentioned in the limitations section, we need to validate our measurements in a different cohort to see how the predictive algorithm correlates with STR. However, STR during percutaneous coronary intervention is recognized as a predictor of the no-reflow phenomenon (3,4). In the no-reflow cases, distal embolization of the plaque or thrombus from the lesion site is a likely mechanism (5,6). Therefore, distal embolization of the plaque or thrombus from the lesion site induced by stent deployment is the probable cause of STR. Based on these data, we believe that STR occurring immediately after stent implantation reflects distal embolization induced by stent implantation.

We had mentioned in the limitations section about the presence of residual luminal thrombus and the ability of the 20-MHz intravascular ultrasound catheter to assess the plaque component. Moreover, we did not have any cases with side branch occlusion in the 11 STR cases, and it should have been included in the exclusion criteria of our study.

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